

Chapter 37

Workup of Chronic Kidney Disease



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Learning Objectives

1. Use an anatomical framework to build a differential for causes of chronic kidney disease.
2. Choose appropriate tests to workup chronic kidney disease.

Clinical Vignette: A 58-year-old man establishes care in your clinic. You notice that his creatinine is 1.8 mg/dL and eGFR is 40. In looking through records, you see that 6 months ago his creatinine was 1.7 and 2 years ago it was 1.4. His past medical history lists hypertension, type 2 diabetes mellitus, and low back pain. No other records are available.

A. Does this patient have CKD? How is CKD defined?

Write down the definition of CKD as shown in Fig. 37.1.

Teaching points

- CKD is defined as daily urine albumin excretion of ≥ 30 mg OR eGFR < 60 mL/min/1.73 m².
- Abnormality must have been present for at least three months.
- This is a continuum: most causes of CKD also have a corresponding version of AKI; therefore, the “definition” is a somewhat arbitrary cutoff.

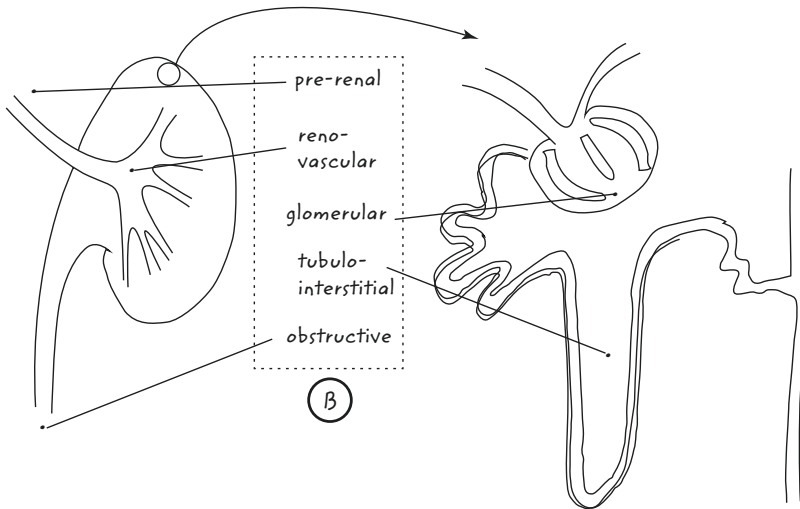
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(A) CKD = $\frac{\text{albumin excretion} \geq 30\text{mg}/24\text{h or GFR} < 60}{> 3 \text{ months}}$



(B) Site	(C) Causes	(D) tests
pre-renal	CHF, cirrhosis, ATN	echo, LFT'S, (mainly history)
renal vascular	RAS/atherosclerosis, fibromusc. dysplasia	Renal US/duplex, CT angiogram
glomerular	hypertensive nephrosclerosis, diabetic nephropathy, nephrotic/nephritic syndrome	UA, spot protein/creatinine
tubulo-interstitial	PC KD, nephrocalcinosis, autoimmune, meds/toxins	Urine micro, autoimmune serologies
obstructive	prostate, bladder, other pelvic	CT, US

Fig. 37.1 Workup of chronic kidney disease: A–D

- B. It is useful to consider the potential causes of his CKD by taking an anatomical approach. What are the five major anatomical sites that we should be familiar with in considering the cause of his CKD?**

Draw the schematic of the kidney and the nephron as shown in Fig. 37.1. Label the sites as the learners list them, and list them in the table at the same time.

- C. What are some specific causes of CKD that correspond to each anatomical site?**

List causes as suggested by the learner.

Teaching points

- Acute tubular necrosis (ATN) can resolve versus progress to CKD.
- Nephritic and nephrotic syndromes start as AKI; some can resolve versus progress to CKD.
- While many of these etiologies have acute correlates, some are truly chronic—such as hypertensive nephrosclerosis, polycystic kidney disease.

- D. The patient’s history will usually guide the work-up for CKD. What initial work-up would you do for this patient? What if the other anatomical sites were suspected?**

List the tests under “glomerular,” and then fill out the rest of the table as tests and studies are suggested by the learners.

Teaching points

- The history is the primary guide for the work-up of the patient.
- If after an initial work-up the etiology is unclear, and especially if the renal dysfunction is worsening, renal biopsy may be needed (as well as nephrology consultation).

Return to Objectives and Emphasize Key Points

1. Use an anatomical framework to build a differential for causes of chronic kidney disease.
 - Return to the diagrams and circle pre-renal, reno-vascular, glomerular, tubulointerstitial, and obstructive.
2. Choose appropriate tests to work up chronic kidney disease.
 - Highlight the common initial tests: urine protein/albumin, UA, abdominal computed tomography (CT) or US, assessment of renal arteries.
 - Renal biopsy may be needed if these tests do not provide a diagnosis or renal failure progresses.

Resources

1. Visconti L, Cernaro V, Ricciardi CA, et al. Renal biopsy: still a landmark for the nephrologist. *World J Nephrol.* 2016;5(4):321–7. <https://doi.org/10.5527/wjn.v5.i4.321>.
2. Fraser SD, Blakeman T. Chronic kidney disease: identification and management in primary care. *Pragmat Obs Res.* 2016;7:21–32. <https://doi.org/10.2147/POR.S97310>.
3. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int Suppl.* 2013;3:1–150.